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A CASE OF APHASIA AND RIGHT HEMIPLEGIA,

With Temporary Spasmodic Conjugate Deviation of the Eyes, Excited by Attempts to Converge the Eyes Strongly towards the Middle Line.

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By Sheridan Delépine, M.B.,
Professor of Pathology, Owens College .

I.—*Preliminary Remarks.*

I am indebted to Dr. W. Ewart, of St. George's Hospital, London, for permission to publish this case, and to use his clinical notes, and to Dr. Richard Sisley, who was at that time Registrar, and has also furnished me with additional notes of great interest. The clinical details, with the exception of those I observed myself, have been abstracted from the notes which these two gentlemen have so kindly placed at my disposal.

Early in May, 1889, Dr. Sisley was showing me some interesting cases in the wards, and we came to the paralysed patient whose case I am about to relate. The extent of the paralysis made me wonder how far forward the softening had extended in the frontal lobe. In a previous case of actinomycotic abscess of the brain,[1] I had noticed a connection between dilatation of the right pupil and affection limited to the hindmost part of the head area.

II.—*Special Observations.*

In this case the head area was certainly much implicated. There was no dilatation of the right pupil. There being no evidence of dilatation or contraction, I tried to see whether there would be any irregularity in the mode of contraction and dilatation of the pupil during accommodation. In order to stimulate the two eyes to the same extent, I moved my finger in the mesial plane from a distance of about three or four feet towards the root of the nose of the patient. The patient followed my finger with both eyes, nothing remarkable being observed till the finger got to about ten to six inches from the eyes, when suddenly the right eye, which till then had converged in the same way as the left, turned to the right, and its axis became parallel to that of the left. The movement was strikingly sudden. I was so taken by surprise that I forgot to observe the pupil. I repeated the observation two or three times, and the phenomenon recurred each time.

[1] *Trans. Path. Soc.*, 1889.

In addition to this, I noticed a remarkable degree of œdema of the right hand. This was so marked when the two hands were side by side, that I suggested that a photograph should be taken of them as a good example of Laycock's nervous œdema. The patient, however, got worse soon after; I never had an opportunity of seeing her again. Dr. Sisley repeated my experiments, and tells me that the conjugate deviation I had observed could not always be produced, but, as will be seen by the notes, fits occurred soon after this, during which there was conjugate deviation of the eyes towards the right.

III.—*Clinical History of the Case.*

The patient was a married woman, aged 23; she had had two children. Seven years before death she had, it appears, an attack of rheumatic fever. Six months before the fatal event she seems to have had another attack. Her left and then her right side became helpless from pain, but she was not paralysed. Four months and a-half before death, after dressing herself without help, she was being driven to the hospital in a cab, when she suddenly exclaimed to her husband who was with her, "Oh, dear, how the cab shakes!" She did not speak afterwards, but nodded her head in answer to questions.

On admission on January 16th, 1889, she was found to have right hemiplegia, face, arm, and leg being quite paralysed; the tongue protruded towards the right; she was aphasic but sensible; the right knee-jerk was increased; there was a loud systolic mitral murmur; the temperature was 101.5° F. She continued for several days in the same state, there being, however, marked improvement in her mental condition, the patient understanding quite well and trying to speak. She seemed to have great pain in the right shoulder and elbow; she had no control over the bladder, but the sphincter was otherwise acting normally for there was no dribbling.

On January 28th she complained of pain in the thorax, and on the following day she had severe rigors with universal shivering and great pallor; the temperature rose to 102.5°; the tendon reflexes were greatly increased on the right side. On February 12th the temperature rose again to 102.5°, and on February 13th hyperæthesia and persistent ankle-clonus of both legs were noticed. On February 17th it was noticed that the patient could say a few words and understood everything quite well; she complained of pain on the top of the head.

On March 1st—about three months before death—the temperature reached 103.5°, the highest point attained whilst the patient was in the hospital; the fingers of the hand began to be permanently flexed (beginning of late rigidity). Between March 12th and 20th great improvement was observed. The patient was quite intelligent; she understood spoken and written language; she could speak better, though her vocabulary was limited and one word was often used for another; she could write after a fashion with her left hand; the sensations appeared to be normal; there was no evidence of pain or tenderness anywhere; the patient could stand with a little assistance.

On March 25th there was a little stiffness on the left side of the neck, and on March 30th it was noted that the right arm and leg, powerless and wasted, were sweating profusely; rigidity was well marked in them. On April 5th—about two

months before death—the patient began to vomit, and from that time had at intervals repeated and severe attacks of bilious vomiting.

On May 3rd—about one month before death—I saw the patient with Dr. Sisley. The general symptoms of hemiplegia with aphasia were very clearly marked, and in addition I noticed the symptoms already described, namely, conjugate deviation of the eyes towards the paralysed side and œdema of the right hand. I found the patient quite intelligent; she understood questions clearly, but could only answer "Yes" or "No." She tried to say things and pointed at objects. She could sit up, and even tried to extend her flexed fingers, but without any result.

On May 14th—three weeks before death—she had two convulsive fits, during which the mouth twitched and the eyes were turned and fixed towards the right (conjugate deviation towards the paralysed side); the lips were pale and the heart very quick. Another similar fit occurred on May 15th, with convulsive motions of the left hand and of both sides of the face. The patient was unconscious for three minutes.

On May 29th it was noted that there had been enuresis for several days. The patient vomited on May 31st; though worse, she could say "Miss Wilson." On June 1st she had two fits, and on June 2nd three fits. She was comatose during the greater part of June 3rd, but was still able to put her arm round her mother's neck. The habitual position of the eye was towards the right. She died on June 4th.

The temperature was nearly always slightly above the normal, being usually 99° to 100° in the morning and 100° to 102° in the evening, and on several evenings rose a little higher, as mentioned above.

IV.—*Necropsy.*

Thirty hours after death the following lesions were noticed in the heart, lungs, brain, spleen, and kidneys. There was extensive disease of the mitral valve, with the usual consequences. The lungs were œdematous, and in a state of brown induration. The spleen and kidneys contained infarcts, some of which were old and cicatrised.

Brain and Spinal Cord.—On opening the cranial cavity an extensive area of softening was observed, affecting the following parts on the upper and outer aspect of the left cerebral hemisphere, posterior parts of the upper, middle, inferior, and ascending frontal convolutions, and also the ascending parietal convolution: The supramarginal convolution and the superior parietal lobule were at most touched by the area of well-marked softening. On the mesial aspect the marginal convolution and gyrus fornicatus were affected over an area corresponding exactly from before backward to the posterior part of the upper frontal convolution, and the upper end of the ascending frontal and parietal convolutions. The appearance of the softened area was very striking, and contrasted very clearly with that of the surrounding brain, so that its limits were not difficult to ascertain. The whole area was much depressed, and very soft to the feel. The convolutions were much shrunken and tortuous; their original shape and arrangement could not be well recognised, but they formed ridges the nature of which could not be mistaken, and which were distinctly continuous with the neighbouring unaltered convolutions. Several

vessels crossed the softened area, and their walls were of a rusty colour, as if stained with broken-up blood pigment. They differed from other vessels, and had evidently contained altered blood. The most posterior of these vessels extended along the anterior and inferior part of the interparietal fissure, but higher up turned forward across the fissure of Rolando towards the inner part of the ascending frontal convolution (this vessel would therefore seem to be the third long branch of the middle cerebral, counting these branches from before backwards), the end of which would have had an unusual direction in this case. These points having been ascertained, the brain being extremely soft had to be slightly hardened before further attempts could be made at further investigation. The distribution of the lesions suggested some irregularity in the blood supply of the part. To dissect the

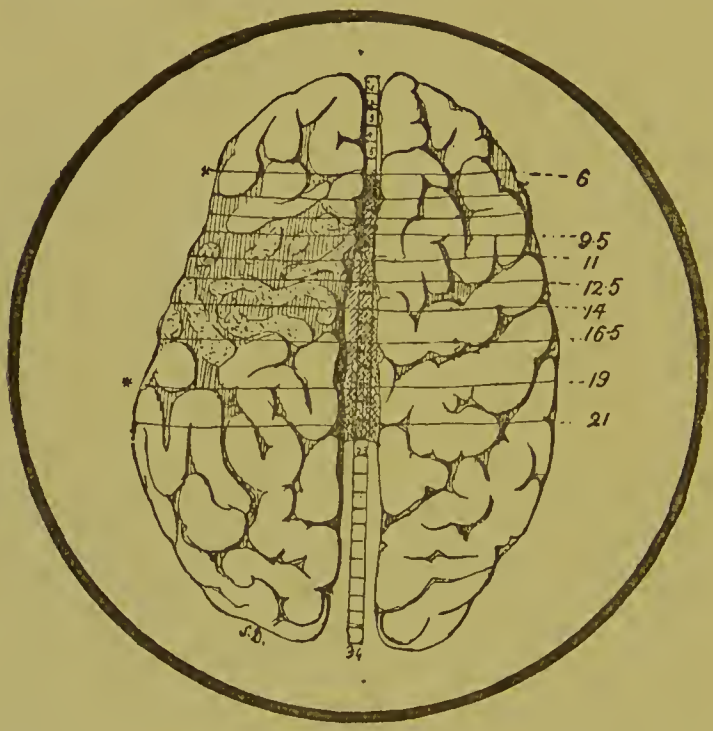

Fig. 1.—Upper aspect of the brain immediately after removal from the cranial cavity. The transverse lines indicate the planes of sections described in the paper. In the middle line is the ½-centimetre scale by which the level of each section is indicated. The asterisks indicate the extreme limits of softening anteriorly and posteriorly. Drawn from Nature by the author.

vessels would, however, have made it impossible to get transverse sections which, owing to the state of the brain, were extremely difficult to obtain and still more to preserve. This anatomical point was, therefore, sacrificed to other more important ones.

In the transverse sections the nature of the lesion was, if anything, more marked than they appeared in the more superficial examination.

The sections were cut at the levels indicated by the trans-

verse lines on the drawing, showing the upper aspect of the cerebral hemispheres.[2]

Only four of the transverse sections made have been photographed, because they were the most perfect and typical. But the whole brain was cut from before backwards in slices seldom more than 1 cm. thick, and often less.

There was no evidence of any degeneration process in front ot the line of section 6, or behind section 19. But the area of complete degeneration extended between the lines 8 to 17 only, that is, did not measure more than $4\frac{1}{2}$ cm. or 5 cm. from before backwards. This is absolutely true of the extent of grey matter affected, but at the posterior limit of the area of degeneration, that is, at the level of the section 16.5,

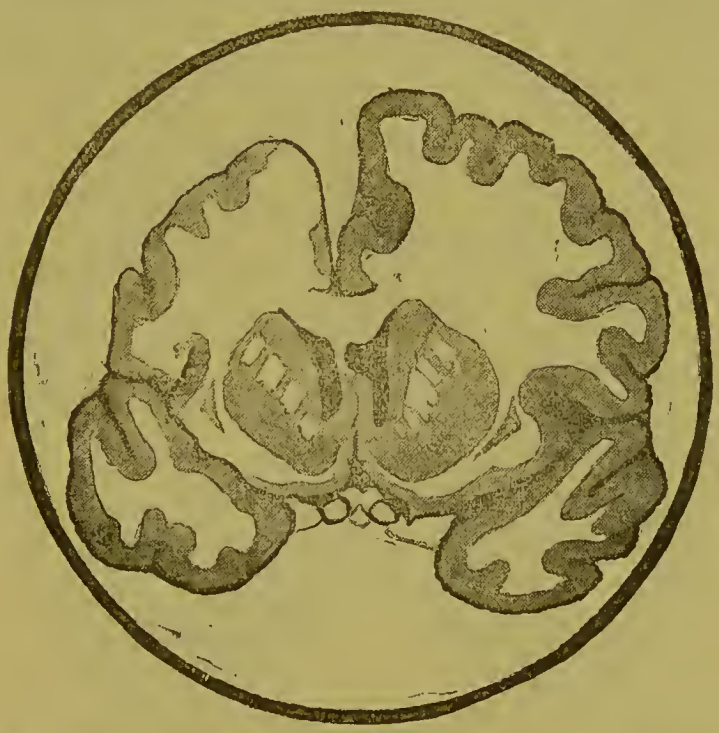

Fig. 2.—Section 9.5, passing between the posterior and middle third of the superior, posterior fourth of the middle, and posterior part of the inferior frontal convolutions, anterior level of the internal capsule, etc. The left hemisphere completely softened at that level; degeneration of cortex not so far advanced as in following sections. The softening extends as far as the middle of the corpus callosum (a faint line indicates the limit). Drawn by the author.

although the grey matter did not seem altered, the white matter was softened at some distance from the surface, nearly as far back as the line 19, that is, the upper end of the ascending parietal convolution.

Photograph 2 (Fig. 2) shows the section 9.5, passing through

[2] The half-centimetre scale placed in the middle line allows the level of the sections to be indicated by numbers. These numbers indicate the distance at which the section was, from the anterior end of the frontal lobes, 6 means. 6 half-centimetres, namely, a frontal section 3 centimetres behind the anterior end of the brain. A half-centimetre scale has been selected, because even a fresh brain can be divided in sections of that thickness.

the posterior third of the superior frontal, posterior quarter of the middle frontal, and the posterior end of the inferior frontal in the region of the inferior part of the pre-central sulcus. The corresponding parts of the marginal convolution and gyrus fornicatus are also cut through. This section passes also through the anterior part of the anterior limb of the internal capsule. All the convolutions named and the white matter under them, as far as the lenticular and caudate nuclei, are destroyed by degeneration, and are considerably atrophied. The softening clearly extends as far as the middle of the corpus callosum; further than that, it is difficult to say whether there was any degeneration or not, owing to the state of the brain at the time of the necropsy. It is, however, to be noted that in one of the sections (14) a small spot of de-

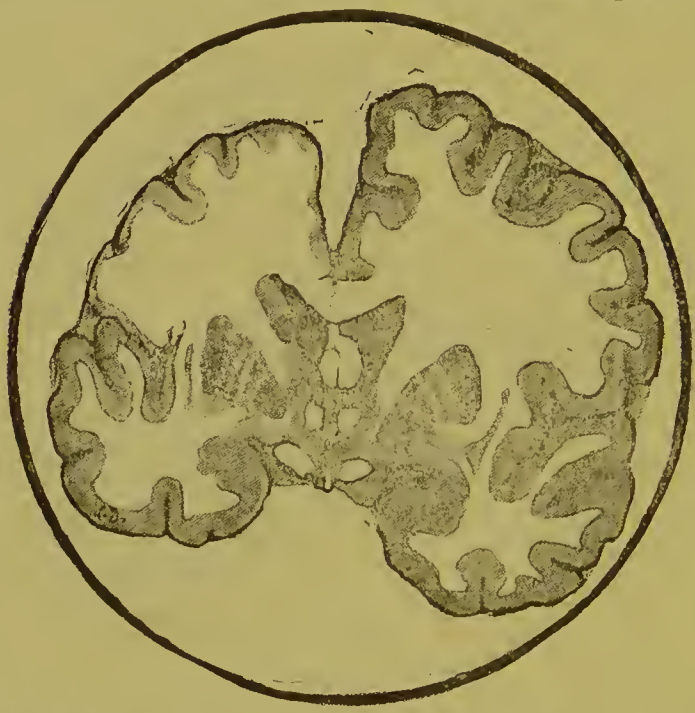

Fig. 3.—Section 12.5, passing through the posterior part of th superior frontal, middle of ascending frontal, and lower end of ascending parietal, also through the knee of the internal capsule and the foramen of Monro. The anterior commissure is seen between the anterior pillars of the fornix. The degeneration is as extensive as, but more advanced than, in Section 9.5. Drawn by the author.

generation, not measuring more than one-sixteenth of an inch in diameter, was found in the very centre of the white matter on the apparently sound side.

*The third photograph** is that of section 11. It passes through the posterior part of the two upper frontal convolutions, the inferior part of the ascending frontal and the corresponding parts of the marginal convolution and gyrus fornicatus. It passes also through the anterior commissure. The extent of the degeneration is the same as in the previous case.

The fourth photograph (Fig. 3) is that of section 12.5. It passes through the posterior end of the superior frontal, the middle

of the ascending frontal, and the lower end of the ascending parietal and the corresponding parts of the median convolutions. It passes also through the knee of the internal capsule and the foramen of Monro. The extent of the degeneration is the same as in the two last sections.

The fifth photograph (Fig. 4) is that of section 14. It passes through the upper half of the ascending frontal and the lower half of the ascending parietal and corresponding parts in the median aspect of the hemisphere.

The section also passes through the anterior part of the optic thalamus, in the region of the middle commissure through the tail of the caudate nucleus and the middle part of the lenticular nucleus it passes through the anterior part of the posterior limb of the internal capsule. The degeneration is as extensive as in the previous sections, a small hæmorrhagic cyst with walls covered with hæmatoidin is visible in the midst of the softened white matter. The section not being

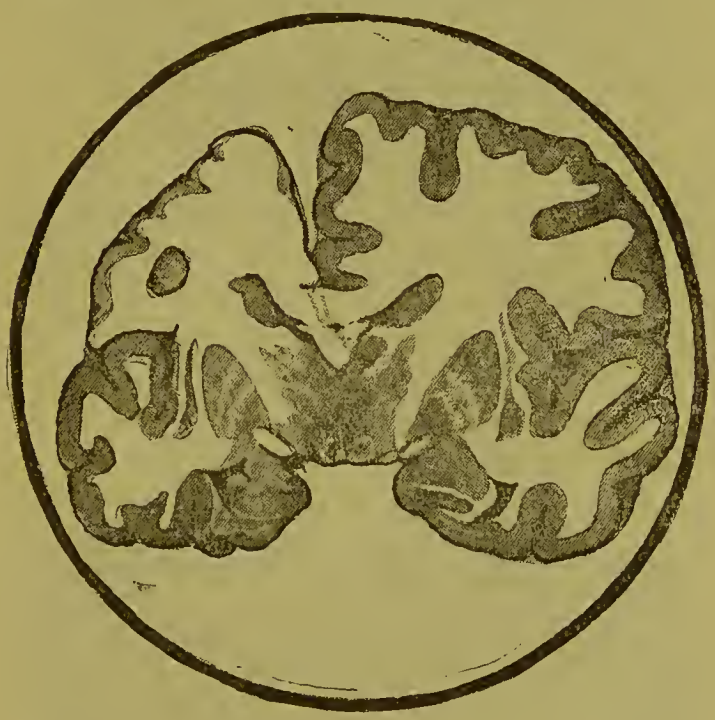

Fig. 4.—Section 14, passing through the upper half of the ascending frontal and the lower part of the ascending parietal, also through the middle commissure and anterior third of the posterior limb of the internal capsule. The degeneration is as advanced and extensive as in Prep. 12.5. A small blood cyst, with walls covered with crystals of hæmatoidin, is in the midst of the softened white matter. Drawn by the author.

stained as the following ones the secondary degeneration of the fibres of the internal capsule cannot be demonstrated.

Other photographs (Figs. 5, 6, 7) have been taken from sections, stained by Weigert's original hæmatoxylin method, and show the secondary degeneration of the fibres of the pyramidal tract in the crusta of the left crus, the pons, the medulla oblongata, and the spinal cord at various levels.

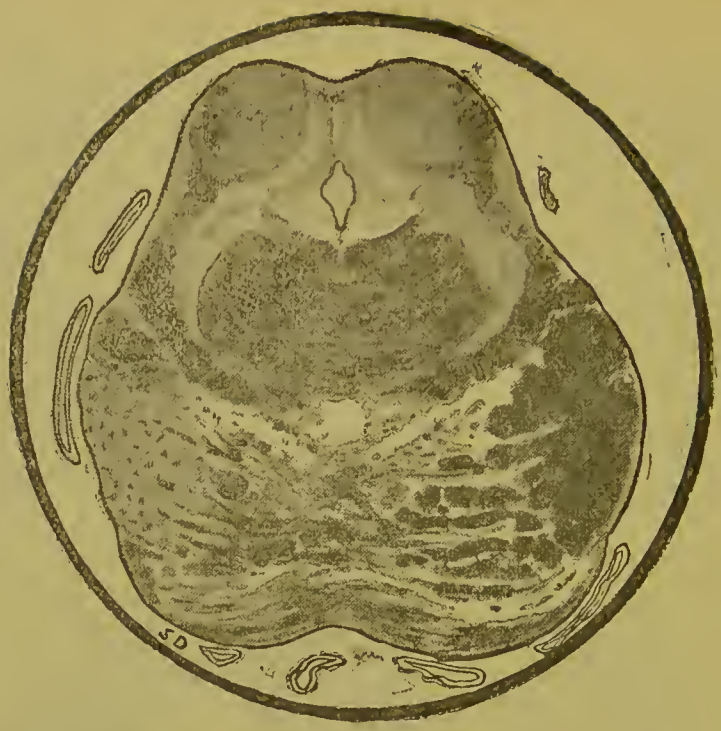

Fig. 5.—Transverse section through the crura cerebri as they pass into the pons, also through the posterior corpora quadrigemina. The greater part of the pyramidal tract (chiefly the middle two-thirds) completely degenerated on the left side. Drawn by the author from a section stained by Weigert's method.

Fig. 6.—Transverse section through the upper half of the pons Varolii, superior and middle cerebellar peduncles, the fillet, etc. Complete degeneration of the left pyramidal tract. Other things can be seen, but are not discussed in the paper. Drawn by the author from a section stained by Weigert's method.

Photograph 6 (Fig. 5) shows transverse sections through the crura cerebri as they penetrate under the superficial transverse fibres of the pons, that is, a little behind the superficial origin of the third. The section passes through the posterior pair of the corpora quadrigemina. The degeneration seems more extensive at this level than at a higher level. This is partly due to imperfect differentiation by the staining. The atrophy of the crus is very evident.

Photograph 7. Transverse section through the pons above its middle. The degeneration of the pyramidal tract on the left side is very clearly shown.

Photograph 8 (Fig. 6). Section through the medulla a little above the apex of the calamus scriptorius. The complete degeneration of the left anterior pyramid is well shown.

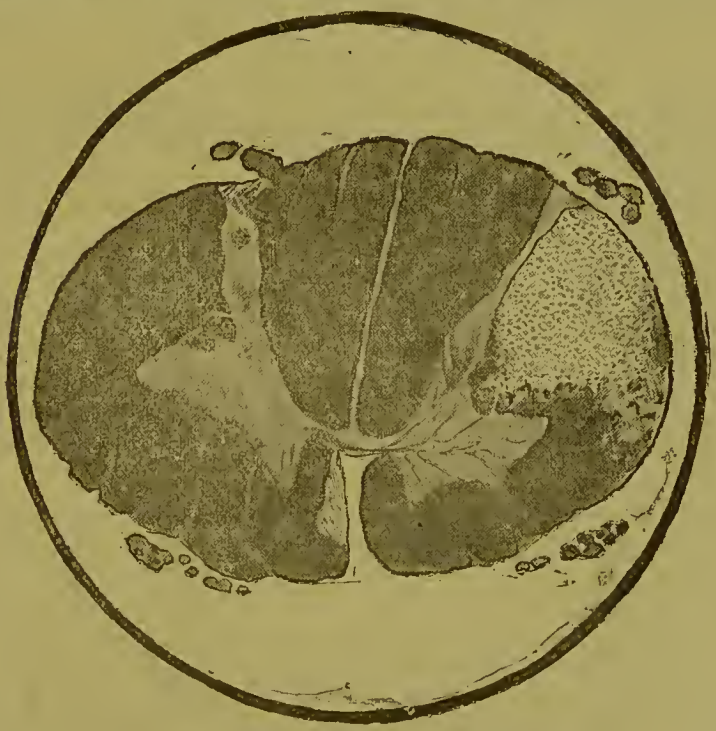

Fig. 7.—Transverse section through the cervical enlargement of the spinal cord at a level between the third and fourth cervical nerves. Secondary degeneration of the right crossed and of the left direct pyramidal tracts; slight extension by secondary inflammation. Drawn by the author from a section stained according to Weigert's method.

*Photograph 9.** Section through the middle of the decussation of the pyramids. One-half at least of the fibres composing the anterior pyramid has passed backwards and towards the posterior part of the right anterior lateral column, and in doing so the diseased fibres become intimately mixed with those of the healthy side. I had thought that in cases where degeneration of the crossed pyramidal tracts on both sides follows a unilateral cortical lesion there might be extension of the sclerotic process by contiguity, so that the fibres of the healthy side might become involved at the decussation. This would account for the occasional occurrence of degeneration of the crossed pyramidal tract on the same side as the lesion without there being recrossing of the fibres. There is no distinct evidence in this case of such an extension, which would,

even if proved, not explain all the appearances described by Sherrington, especially regarding the number of fibres degenerated at various levels.

Photograph 10 (*Fig. 7*). Section through the spinal cord in the cervical region between the third and fourth cervical nerves. The degeneration of the right crossed pyramidal tract and of the left direct pyramidal tract is very clear. At this level the crossed pyramidal tract reaches the surface of the cord behind the direct cerebellar tract.

*Photograph 11.** Section through the cord in the dorsal region (sixth and seventh dorsal nerve). The degeneration of the crossed pyramidal tract is very well marked.

*Photograph 12.**—Sections through the cord in the lumbar region between the third and fourth lumbar nerves. The appearance is very much the same as in the last section.

V.—*Conclusions.*

Putting aside a number of issues which are of great interest, and confining my remarks to the eye symptoms to which I have alluded, I wish to attract attention to the following facts:

1. The patient had extensive degeneration of the motor areas of the brain on the left side.

2. From the distribution of these lesions and from certain convulsive phenomena observed during life, it is safe to conclude that the area of destruction was surrounded by a zone of irritation and inflammation, leading to gradual extension of the area involved.

3. In the crura, pons, medulla, and cord the secondary degenerations were typical, and there seems no evidence of there having been in any of these regions any concurrent process complicating or obscuring the effects of the cortical lesion.

4. About the period when symptoms of inflammatory extension became manifest, that is, one month at least before death, I observed that when the patient tried to converge her eyes towards the middle line so as to see an object placed near the root of her nose, the axis of the right eye was suddenly deviated from the middle line, and became parallel with that of the left, this producing conjugate deviation towards the paralysed side, that is, the same effect which would have been produced by stimulating some parts of the head area on the diseased side of the brain (Ferrier, Horsley, and Schäfer, Horsley and Beevor, Mott and Schäfer).

5. It seems safe to conclude from these premisses that the deviation of the eyes towards the paralysed side was the result of the stimulations of the centre already implicated, though not destroyed by the disease, and that the inflammatory irritation of that centre, added to the stimulation resulting from impulses sent by some higher centre, upset the balance, which would have resulted from the volitional stimulations of both centres. (The experiences of Mott and Schäfer on unilateral and bilateral stimulations of the head and eye areas in the monkey are extremely important in connection with this).

6. The phenomenon being produced at the time when I observed it first, only under the influence of a voluntary effort, the effect of volitional impulses can be compared with those of a physical stimulus such as that of an electrical current. Whether this be due to direct nervous impulses or vasomotor changes in the area cannot be established in this case.

7. Later on, when the irritative process had become more intense, the deviation was produced without the additional stimulus of a voluntary effort.

8. However important the bearings of phenomena observed in a single case may be, it is difficult to speak of their practical value; it seems, however, probable that in a certain number of cases the same phenomena will be observed, and that they may give additional help in the diagnosis of the localisation of some lesions.

(*Note.*—To secure clearness, the photographs have not been reproduced directly, but have been carefully outlined by the author. The photographs themselves were demonstrated at the meeting. Those marked with an asterisk have not been reproduced.)